children and adults, such as the one that follows, may be of value.

IgE—units per ml				
Infants	Children and Adults		Percent of the	Allergic
<2 yrs.	>3 yrs. (RIA)	(PRIST)	Population	Percent
<10	<100	<65	68	<5
10-20	100-350	65-230	20	20
21-100	351-750	231-500	9	35
>100	>750	>500	<3	~100*

*In the absence of helminthiasis.

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Occupational Asthma

OCCUPATIONAL ASTHMA refers to a disease in those persons in whom asthma develops primarily from exposures at their work or hobby sites. Although occupational asthma dates at least to Ramazzini's reports in 1700 of asthma provoked by flour dust in bakers and wig makers, the sources of exposures seem to be on the increase in recent years. The provoking agents may be divided into those of (1) *animal origin* affecting poultry farmers; veterinarians; horse, cattle and sheep breeders; chicken inspectors; furriers, and aviarists; (2) *vegetable origin:* farmers, hempworkers, grainworkers, bakers, millers and woodworkers; (3) *inorganic chemicals:* chromium, platinum, aluminum and nickel factory workers, electricians, welders and solderers, and (4) *organic chemicals:* plastic makers and painters who handle isocyanates; meat wrappers exposed to polyvinyl fumes; workers in pharmaceutical plants, and workers using enzymes such as trypsin, Bacillus subtilis enzymes and papain. These are but a few examples of an almost endless list of occupations and causative agents in occupational asthma.

Asthma may occur not only in those who are predisposed by being atopic but also in apparently normal persons without a personal or family history of allergies or IgE specific antibodies. There is some evidence that the atopic persons have a more rapid onset of symptoms following exposures and tend to leave their jobs sooner. Asthma may occur within minutes of a sufficient exposure and responds well to bronchodilators. Others may have asthma delayed several hours after exposure and are better controlled by steroids than by epinephrine or theophylline. Some experience both immediate and late onset asthma.

The pathogenic mechanisms are not always clear-cut. Some agents such as animal danders and enzymes are allergens producing specific IgE antibodies even in nonatopic persons. Others such as polyvinyl and acetylene fumes are respiratory irritants, while still others like toluene diisocyanate (TDI) may have both antigenic and irritant properties.

The examining physician can be alerted to the relationship of asthma to job or hobby by obtaining a careful history. A tip-off is the waxing and waning of symptoms, worse during workdays and better on holidays, weekends and vacations. Sometimes the exposure source may not be readily apparent, such as the case of a secretary whose office is clean but who shares access or ventilation with an adjoining factory. Confirmation that the suspected agent does provoke asthma may require bronchial challenge and immunological studies on the patient and control subjects. Discovery of occupationally induced asthma is rewarding, since removal from the exposure may well effect a cure.

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Emergency Room Treatment of the Adult Asthmatic

DURING THE COURSE of chronic asthma, acute attacks occur that can be effectively treated in a hospital emergency room. A brief initial history can determine the possible precipitating cause of the attack, such as exposure to allergens or respiratory irritants, intercurrent viral respiratory infections, aspirin or other analgesics, emotional trauma, inadequate maintenance therapy, abrupt withdrawal of steroid medications, or overuse of aerosol bronchodilators.

Severe bronchial obstruction produces decreased breath sounds, notably prolonged expiration, use of accessory muscles of respiration and pulsus paradoxus. Tachycardia and mild hypertension are characteristic of acute asthma and not necessarily complications of treatment. Cyanosis, quiet chest, restlessness and exhaustion are ominous signs of impending or actual respiratory failure

Arterial blood gas measurements are essential